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# The mechanisms operation of thermodynamic system of a human organism

**Ponizovskiy Michail**

Kiev, Ukraine, "Kiev regional p/n hospital", /Head of "Laboratory Biochemistry and Toxicology"/, 08630 Kiev region, Vasilkov district, Glevaha, Voksalna 8, Ukraine

**Email address:**
ponis@online.de

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**Abstract:** Studying the open non equilibrium non linear thermodynamic system of an organism there were explained mechanisms maintenance stability Internal Energy and Internal Medium as an organism as well as cells an organism in norm and in quasi-stationary pathologic states from the point of view of thermodynamic laws. There were calculated the main thermodynamic parameters according thermodynamic laws of open non equilibrium non linear thermodynamic system of a human organism. Also it was explained thermodynamic mechanisms stability open non equilibrium non linear thermodynamic system of a human organism, according Prigogine theorem, as well as development open non equilibrium non linear thermodynamic system of a human organism, according Glansdorff and Prigogine theories. There were explained the mechanisms of mutual interactions and interdependence between thermodynamic system of an organism and thermodynamic systems of cells of an organism for maintenance stability Internal Energy both an organism and cells of an organism. Besides there were described mechanisms operation of cellular capacitors contributing to maintenance stability Internal Energy both an organism and cells of an organism. Moreover there were described remote cellular reactions via cellular capacitors operations which are preceding contacts cellular reactions leading to immune reaction on strange objects into an organism that saves stability Internal Energy and Internal Medium of an organism. Also it was described interdependence between nuclear capacitors, mitochondrial capacitors, intracellular organelles capacitors and cellular capacitors for maintenance stability Internal Energy both an organism and cells of an organism. Besides there were elucidated oscillating interdependence between anabolic and catabolic processes both in nucleus and mitochondria leading to cells development via cellular cycle.

**Keywords:** Thermodynamic System of an Organism, Thermodynamic Laws, Internal Energy, Entropy, Enthalpy, Stationary State of an Able-Bodied Organism, Three Levels of an organism's regulative system

## 1. Introduction

The mechanisms maintenance stability Stationary State of open non-equilibrium non-linear thermodynamic system of an able-bodied organism and also Quasi-stationary State of open non-equilibrium non-linear thermodynamic system of a sick organism are exerted by three levels of regulative mechanism: highest level regulation [CENTRAL NERVOUS SYSTEM], high level regulation ["Equilibrium Constant of ionic metabolism", "Equilibrium Constant of acid –- alkaline metabolism", "Equilibrium Constant of oxidative – reduction Potentials of metabolism" and "Equilibrium Constant of coagulating system of blood"] and low level regulation ["Equilibrium Constant of energy exchanges" and "Equilibrium Constant of metabolism"] [1, 2]. Also cells from all sections of an organism (blood, lymph, neurolymph, tissues) are connected with one another due to remote reactions across distance as the results of their cellular capacitors operation via resonance waves, maintaining common stability of Internal Energy both in cells and in an organism creating activity of immune defensive system and autophagy [3]. The mechanism maintenance stability Stationary State of open non-equilibrium non-linear thermodynamic system of an able-bodied organism was explained using Prigogine theorem.

# 2. Material and Methods

The structure of an open non equilibrium non linear thermodynamic system of a human organism and regulatory mechanisms are the Material investigation of the article. The thermodynamic mechanism maintenance stability Internal Energy and Internal Medium of Stationary State in norm and in Quasi-stationary States [pathologic states] of an open non equilibrium non linear thermodynamic system of a human organism is the Methods investigations of the article.

### *2.1. Regulation Processes Promoting Maintenance Stability of Internal Energy and Internal Medium*

The maintenance stability of Internal Medium (constant concentration substances in blood and in neurolymph) and Internal Energy (stable temperature 36.0°C – 36.9°C by which all enzymes operate etc.) both in an organism, and in each cell of an organism, occurs via low level of regulation due to moderate fluctuating shifts of the balance anabolic endoergonic and catabolic exoergonic processes either in anabolic pathway or in catabolic pathway [3, 4]. Also the low level of regulation is subjected to influence of Environment and affects mutually on Environment. There are also the mutual interactions between low level of regulation and high level of regulation [1, 2, 4, 5]. The CENTRAL NERVOUS SYSTEM, as the highest level regulation, affects both on high level of regulation and on the low level of regulation [1, 2]. The regulation biophysical and biochemical processes in an organism influences on regulative processes in cells creating maintenance stability cellular Internal Energy and cellular Internal Medium via interactions between extracellular ($\mu_{extcell}$) and intracellular chemical potentials ($\mu_{intcell}$) that induces operations of cellular capacitors. Also stability cellular Internal Energy and cellular Internal Medium are supported by interactions as between cellular capacitors of cellular wall and intracellular capacitors (nucleus capacitors, mitochondria capacitors and capacitors of all organelles) as well as between cellular capacitors of all cells and substances in surrounding medium of cells in an organism.

There are the important footnotes:

*Hormones cause influences on cellular processes and are the links in all level Regulations: Highest level Regulation, High level Regulation and Low level Regulation.

*Enzymes operate in biochemical reactions of all level Regulations: Highest level Regulation, High level Regulation and Low level Regulation.

### *2.2. Mechanism Maintenance Stability Internal Energy in the Open Thermodynamic System of an Organism [3]*

The periodicity of rhythmic activity cellular processes of exocytosis and endocytosis promotes stability Internal Mediums and Internal Energy as in cells as well as in an organism. The consumption and accumulation of energy in Internal Mediums of cells via intensive anabolic endoergonic processes resists to dissipation energy via catabolic exoergonic processes (i.e. entropy) that promote minimization gain of entropy according to Prigogine theorem and contribute to maintenance of stability open thermodynamic systems both in cells and in an organism [3].

Here is the theorem Prigogine:

[The symbols: Entropy – S; Stream of Substances – Js; Stream of Energy – Je; Force of Substances – Fs; Force of Energy – Fe; Phenomenological Streams of Substances and Energy – Zss and Zee]

$dS = JsFs + JeFe > 0$. Conjugated flows:

$$Js = ZssFs + ZseFe \text{ and } Je = ZeeFe + ZesFs.$$

$dS = (ZssFs + ZseFe)Fs + (ZeeFe + ZesFs)Fe = ZssFs^2 + ZseFsFe + ZesFsFe + ZeeFe^2 = ZssFs^2 + 2ZseFsFe + ZeeFe^2 > 0$. Corresponding to the Onsager concept:

$$Zse = Zes.$$

However there are $Zss > 0$; $Zee > 0$; $Zse > 0$.

We can conceive that there is not change flow of Substances in Stationary State, i.e. $Js = 0$. Just after all, the concentrations of Substances in Internal Medium of an organism (in blood and in neurolymph) are constant, i.e. the quantity inflow of the Substances into Internal Medium of an organism is equal the quantity outflow of the Substances from Internal Medium of an organism. Hence there is the derivative dS from Fs, if it's Fe = const (the constant production calories for maintenance temperature 36.0ºC – 37.5ºC by which all enzymes operate):

$$dS/dFs = 2\,ZseFe + 2\,ZssFs = 2\,(ZseFe + ZssFs) = 2\,Js = 0.$$

The second derivative (flexon) from S is peer: $d^2S / dFs^2 = 2Zss > 0$

It corresponds to extreme point. It means that $dS \rightarrow \min$.

So the minimization increment of dissipation energy via minimization of gain entropy proves the stability of the open non equilibrium thermodynamic system of an organism.

Thus the balance between catabolic and anabolic processes defines chemical potentials ($\mu$) which are driving mechanism as for transport substances across cellular membranes as well as for maintenance stability of Internal Energy and Internal Medium of an organism that correspond to first law of thermodynamics.

### *2.3. First Thermodynamic Law in Thermodynamic System of a Human Organism*

According to first law of thermodynamics [$Q = \Delta U + W = \Delta H$], Stationary State of open non-equilibrium non-linear thermodynamic system of an able-bodied organism and also Quasi-stationary State of open non-equilibrium non-linear thermodynamic system of a sick organism are characterized by stability of Internal Energy ($\Delta U$) (stable temperature 36.0°C – 36.9°C by which all enzymes operate, stable PH = 7.35 in blood etc.). The stable Internal Energy

(ΔU) promotes Stability of Internal Medium of an organism, i.e. stability concentrations of substances in blood and in neurolymph. The stability of Internal Energy (ΔU) is maintained by Common Works (W) which consists of Life Work ($W_{life}$) [metabolic processes, heart work, blood circulation, muscles activity, cellular activity and so on] and Active Work ($W_{act}$), causing mutual influences between Environment and human activity. Total Heat Energy (Q) is presented by Enthalpy (ΔH) which promotes development of an organism due to oscillation Energy as positive fluctuation entropy (+Δβ), according to Glansdorff and Prigogine theory [1 – 5].

### *2.3.1. The Calculation of Entropy an Organism*

The stable pH = 7.35 in blood is the one of the main indices of stable Internal Energy in Stationary State of an organism. The Life Works of an organism maintain stability pH = 7.35 in blood of an organism for maintenance stability an organism's Internal Energy via regulative mechanism of an organism [1, 2, 4]. The basic meaning of acid – alkalinity metabolism is expressed by the Equilibrium Constant of hydrocarbonaceous buffer ($K_{buffer}$) which is the main buffer of "Equilibrium Constant of acid –- alkaline metabolism" and is calculated by the Henderson – Hasselbach formula:

$$K_{buffer} = \frac{[H_3O^+][HCO_3^-]}{[H_2CO_3]} = 4.31\times10^{-7}$$

The Equilibrium Constant of hydrocarbonaceous buffer ($K_{buffer}$) is $4.31*10^{-7}$ that corresponds to $pK_{buffer}$ = 6.35. Molar concentration of hydrocarbonaceous ions in human blood is 20 - 32millimole/l (average index is equal 26millimole/l) in norm. Just the average index 26millimole/l reflects stable Internal Energy in Stationary State of an organism and is the mechanism of the stable pH = 7.35 in blood of an organism.

Having taken the logarithm of the Henderson – Hasselbach formula we receive:

$$\lg K_{buffer} = \lg [H_3O^+] + \lg [HCO_3^-] - \lg [H_2CO_3]$$

$$pK_{buffer} = pH + \lg [HCO_3^-] - \lg [H_2CO_3]$$

$$6{,}35 = 7{,}35 + \lg [26] - \lg [H_2CO_3]$$

$$\lg [H_2CO_3] = \lg [26] + 1.0 = 2.415$$

$$[H_2CO_3] = 260\text{millimole/l} = 0.26\text{mole/l}$$

There is the outcome of the last step of catabolic exoergonic oxidative processes which leads to dissipation Energy ($-2nH^+$) and substances ($CO_2$ and $H_2O$) into environment:

$$0.26\ H_3O^+ + 0.26\ HCO_3^- = 0.26\ H_2CO_3 + 0.26H_2O = 0.26CO_2 + 0.52H_2O$$

This reaction reflects loss of Energy. Just the energy of $CO_2$ – 0.26mole/l reflects loss of Energy of an organism due to interactions between an organism and Environment. Therefore it should be calculated the state of an energy of an organism in a neutrally temperate environment which can been surmounted by organism without supplemental help, i.e. 20°C – 25°C or 293.5°K – 298.5°K in environment, considering stable normal temperature an organism 36°C – 36.8°C or 309.5°K – 310.3°K. The difference between neutrally temperate environment and normal temperature an organism is 11.0°C – 16.8°C or 11.0°K – 16.8°K. The energy of stable normal temperature an organism 36°C – 36.8°C due to balance anabolic and catabolic processes reflects stable Internal Energy of an organism.

However maintenance stable energy for stability of normal temperature an organism 36°C – 36.9°C is impossible out of the levels between neutrally temperature environment and normal temperature an organism, i.e. out of 11.0°C – 16.8°C, because an organism needs of catabolic exoergonic processes as for dissipation energy into environment as well as for maintaining normal temperature 36.6°C of an organism and for transformation the thermal energy into mechanical energy for cardiac muscles actions and vascular muscles actions also into electric energy of neural system actions etc. Environmental Temperature out of the levels between neutrally temperature environment and normal temperature an organism compels an organism either to produce supplementary dissipative energy via intense catabolic processes or to inhibit catabolic processes for maintenance temperature an organism 36°C – 36.9°C.

Thus there is the calculation of an organism's loss Energy [the symbols: $Q_{loss}$ – loss Energy, R – gas constant, M – moles, T – temperature Kelvin]:

$$Q_{loss} = RMT = 8.31\ J/MT^{\ x}\ 0.26^{\ x}\ (11 – 16.8) = 23.77\ J – 36.298\ J = 5.68\ cal – 8.675\ cal.$$

Entropy (S) and thermodynamic Helmholtz potential (F) form Internal Energy (U) [F = U - TS]. Thus Entropy (S) is the index of Internal Energy (U) of an organism which reflects interactions between an organism and Environment. Moreover fluctuation of Entropy (S) exerts development of an organism according Glansdorff and Prigogine theory [15] that depends also on interactions between an organism's Internal Energy (U) and Environment, i.e. difference between neutrally temperate environment and normal temperature an organism as the parameter of Internal Energy (U) an organism - 11.0°C – 16.8°C or 11.0°K – 16.8°K. This difference of temperatures promotes organism's loss Energy ($Q_{loss}$) that leads to Entropy (S) because non whole loss Energy ($Q_{loss}$) can been restored.

Therefore there is the calculation Entropy (S) according to second law of thermodynamics Q = ΔST:

$$\Delta S = (8.67\text{cal} - 5.68\text{cal}) : 310.1°K = 0.0096\ \text{cal}$$

### *2.3.2. Calculation of Internal Energy (U) of an Organism*

Just the Internal Energy of an organism results in stable pH = 7.35 in blood of an organism;

$\lg [H_3O^+] = 7.35$, i.e. $H_3O^+ = 22400000$micromole/l

Thus pH = 7.35 satisfies the requirements of such equation: $[H^+] = 22400000$ micromole/l = 22.4 mole/l.

Thus 22.4 mole/l of $H^+$ are divided between catabolic exoregonic processes and anabolic endoergonic processes that contribute to the stable normal temperature an organism 36°C – 36.8°C or 309.5°K – 310.3°K (the average temperature 36.6°C or 310.1°K) reflecting stability Internal Energy of an organism. The calculation of Internal Energy (ΔU) via hydrogen ions requires to consider thermodynamic characteristic of concentration hydrogen ions 22.4 mole/l, using the Table 1. [6].

***Table 1.*** *Standard chemical potential $\mu_t$°, Standard partial molar enthalpy $h_t$°, and Standard partial molar entropy $s_t$° for the few hydrated ions: Standard state 101.3 KPa, 298°K unit activity in molar scale.*

| Substances | State | $\mu_t$/kJ∗mole$^{-1}$ | $h_t$ / kJ∗mole$^{-1}$ | $s_t$ / JK$^{-1}$∗mole$^{-1}$ |
|---|---|---|---|---|
| $CO_2^{2-}$ | hydrated ion | -527.9 | -677.1 | -56.9 |
| $Cl^-$ | hydrated ion | -131.2 | -167.2 | 56.5 |
| $Fe^{2+}$ | hydrated ion | -78.9 | -89.1 | -137.7 |
| $Fe^{3+}$ | hydrated ion | -4.7 | -48.5 | -137.7 |
| $H^+$ | hydrated ion | 00 | 00 | 00 |
| $Na^+$ | hydrated ion | -261.9 | -240.1 | 59.0 |
| $OH^-$ | hydrated ion | -157.3 | -230.0 | -10.7 |
| $SO_4^{2-}$ | hydrated ion | -744.6 | -909.3 | 20.1 |
| $HS^-$ | hydrated ion | 12.6 | -17.6 | 63.8 |
| $Zn^{2+}$ | hydrated ion | -147.1 | -153.9 | -112.1 |

There is the ions reaction which forms hydrocarbonaceous buffer: $2H^+ + CO_2^{2-} = H_2CO_3$

If Standard chemical potential $\mu_t$ of $CO_2^{2-}$ is assumed equal 00, so Standard chemical potential $\mu_t$° of hydrated ion $H^+$ should be equal 527.9 $\mu_t$°/kJ∗mole$^{-1}$, i.e. it is the coefficient for calculation Energy in kilojoule creating by mole of hydrogen ion $[H^+]$ resulting in stable PH = 7.35. The Energy of hydrogen ion $[H^+]$ moles in stable PH = 7.35 is Internal Energy (ΔU) of an organism.

Thus there is the calculation energy of Internal Energy (ΔU) of an organism:

ΔU = 22.4mole/l $^{x}$ 527.9 $\mu_t$°/kJ∗mole$^{-1}$ = 11824.96kJ = 2826.165kcal = 2826165cal.

Internal Energy (ΔU) includes catabolic thermal energy for maintenance stability temperature 36°C – 36,8°C by which all .enzymes operate, energy of anabolic proliferative processes for maintenance balance catabolic & anabolic processes and for maintenance stability Internal Medium (stable concentration substances in blood and neurolymph), mechanical energy of heart operation for blood circulation, lung operation and muscles operation, chemical and electrical energy of maintenance all Equilibrium Constants of three levels regulation inducing by Central Nervous System etc.

### *2.3.3. The Calculation of Common Work (W) and Enthalpy (ΔH) of an Organism*

Basal metabolic rate (BMR) is Basal Work ($W_{basal}$) which promotes stability Internal Energy (ΔU) of an organism. Basal metabolic rate (BMR), as Basal Work ($W_{basal}$) of an organism, defines the energy at rest of an organism which promotes as maintenance normal function of an organism's vital organs [the heart, lungs, nervous system, kidneys, liver, intestine, sex organs, muscles and skin], as well as mechanism maintenance stability Internal Energy of an organism [1, 2]. Basal metabolic rate (BMR) or Basal Work ($W_{basal}$) shows the production of necessary calories for maintenance of ability of an organism's life in a condition of full rest, i.e. the quantity of the energy, which body spent when person sleeps whole day. Basal metabolic rate (BMR) or Basal Work ($W_{basal}$) also resists influences of an environment for maintenance stability Internal Energy of an organism in the state of its rest. Normal average quantity of Basal metabolic rates (BMR) or Basal Works ($W_{basal}$) are for male 1510cal – 1945cal per day and for female 1243.4cal – 1589.9cal per day using Harris-Benedict Equation.

Work in physical Activity Calories = (BMR + Activity):

Sedentary Active percent 20% (Sitting most of the day), i.e. Activities of Sedentary are for male supplementary calories 302cal – 389cal and for female supplementary calories 248.68cal – 317.98cal.

Thus Sedentary Active Works ($W_{sed.act}$) are for male 1812cal – 2334cal per day and for female 1492.08cal – 1907.88cal per day.

Lightly Active percent 37.5% (Walking here and there; daily chores), i.e. Activities of Lightly Active Actions are for male supplementary calories 566.25cal – 729.375cal and for female supplementary calories 466.275cal – 596.2125cal.

Thus Lightly Active Works ($W_{ligh.act}$) are for male 2076.25cal – 2674.375cal per day and for female 1709.675cal – 2186.1125cal per day.

Moderately Active percent 40% (Constantly moving around; daily exercise), i.e. Activities of Moderately Active Actions are for male supplementary calories 604cal – 778cal and for female supplementary calories 497.36cal – 635.96cal.

Thus Moderately Active Works ($W_{mod.act}$) are for male 2114cal – 2723cal per day and for female 1740.76cal – 2225.86cal per day.

Very Active percent 50% (Heavy exercise for prolonged

periods of time, such as training for a sport), i.e. Activities of Very Active Actions are for male supplementary calories 755cal – 972.5cal and for female supplementary calories 621.7 cal – 794.95 cal.

Thus Very Active Works ($W_{very.act}$) are for male 2265cal – 2917.5cal per day and for female 1865.1cal – 2384.85cal per day.

Normal resistances of Stationary State or Quasi-stationary State of an organism to environmental influences are realized by Physical Activity of Active Work ($W_{act}$), which inserts Active Action Calories to Basal Work ($W_{basal}$) creating Common Work (W). Just it must recognize Moderately Active Work ($W_{mod.act}$) as the Common Work (W).

Thus Common Work (W) shows for male 2114 cal – 2723 cal per day and for female 1740.76 cal – 2225.86 cal per day.

Enthalpy (ΔH) represents general energy providing maintenance stable operation of Internal Energy (ΔU) and stability of Internal Medium.

There are the calculations of Enthalpy (ΔH), using equation $\Delta H = \Delta U + W$, for male [2826165 + 2114 = 2828279 cal] – [2826165 + 2723 = 2828888 cal] per day and for female [2826165 + 1740.76 =2827905.76 cal] – [2826165 + 2225.86 = 2828390.86 cal] per day. Thus Enthalpies are for male 2828.279 kcal – 2828.888 kcal per day and for female 2827.906 kcal – 2828.391 kcal per day.

### *2.4. Mechanisms of Cellular Distance Reactions for Maintenance Stability Internal Energy and Internal Medium of an Organism*

#### *2.4.1. Influence of Chemical Potentials on Electrical Charges of Cellular Membranes [3]*

Effective differentiation both pathological cells and normal cells is carried out by histological methods of a coloration of intracellular medium various cells. Cytoplasm, kern and organelles (mitochondria, lysosomes, ribosomes etc.) of different cells accept different staining agents [different granulocytes (eosinophilic granulocytes and basophilic granulocytes), different agranulocytes (basophilic color of lymphocytes and monocytes), pathological undifferentiated cells, pathological cells granulocytes' row (promyelocytes, metamyelocytes, myelocytes etc.), pathological cells of lymphocytic row (lymphoblasts) etc]. However the intracellular medium of different cells accept either more acidic staining agents or more alkaline staining agents that reflects various chemical potentials of internal mediums (int.μ) of these cells: ($int.\mu_1 \neq int.\mu_2 \neq int.\mu_3 \neq int.\mu_n$ etc.). Chemical potentials of cellular internal mediums influence on electrical charges of inner membranes of cell's walls: ($int.q_1 \neq int.q_2 \neq int.q_3 \neq int.q_n$ etc.). Electrical charges of external membranes of cell's walls reflect chemical potential in surroundings of cells [environment] (env.μ): [$ext.q_1 \approx ext.q_2 \approx ext.q_3 \approx ext.q_n$]. The electrical charges of external membranes of cellular walls are changed a little in these conditions [$ext.q_n \rightarrow ext.q_{n1} \rightarrow ext.q_{n2}$ etc.].

#### *2.4.2. Transports Substances across Cellular Membranes as the Mechanisms Stability of Open Thermodynamic Systems Cells and an Organism*

Driving forces of substances transport through cellular membrane operate by the interaction between the chemical potentials (μ) of intracellular medium and extracellular medium, as well as osmotic pressures (π), concentrations of substances (c), promoting movements of substances that is reflected in formulas of substances transport: Fick formula and Theorell formula [4, 7]. Anaerobic catabolic processes of Glycolysis exert the energy utilization via energy accumulation in lactic acids for advance of anabolic endoergonic processes promoting proliferative processes [7]. The chemical potentials of intracellular medium ($\mu_{intcell}$) are created by chemical potentials ions ($\mu_{ion}$) and ionized molecules of substances ($\mu_{ion.mol}$). The interaction of an intracellular chemical potential ($\mu_{intcell}$) with an extracellular chemical potential ($\mu_{extcell}$) is the driving mechanism of substances transport across cellular membrane [3, 7 – 9]. Destruction of substances by enzymes leads to the increase quantity of ionized molecules within granules in the cytoplasm that increases as osmotic pressure ($\pi_c$) and as well as the chemical potential of intracellular mediums ($\mu_{intcell}$) [3, 8 – 10]. An extracellular medium keeps stability biochemical indices of Internal Medium an organism in which stabile parameters of substances concentrations are maintained (in blood, in neurolymph etc.) [11, 12]. The mechanisms of maintenance the stability of an organism's Internal Medium and Internal Energy (U) promote intensive excretion of metabolic products and dissipation of energy into Environment. The difference between chemical potentials in intracellular mediums ($\mu_{intcell}$) and in extracellular mediums ($\mu_{extcell}$) does not become null because of presence in cellular medium the more intensive anabolic processes than in extracellular medium that maintains the permanent cellular cycle and proliferative processes. Thus the consumption and accumulation of energy in internal mediums of cells via intensive anabolic endoergonic processes resists to dissipation energy via catabolic exoergonic processes (i.e. entropy) that promote minimization gain of an entropy according to Prigogine theorem (see above) and contribute to maintenance of stability open thermodynamic systems both cells and an organism [3 – 5].

#### *2.4.3. Detections extracellular and intracellular Chemical Potentials which induce the Charges on the Cellular Membranes of the Cellular Capacitors*

An extracellular medium /blood plasma/ is visualized by staining agents via histological methods of coloration poorly because of great catabolic processes with great outflow substances and very small anabolic processes in it. On contrary a cytoplasm is visualized by staining agents via histological methods of coloration into clear basophilic color, accepting alkaline staining agents, i.e. having bonds to negative ionized molecules or covalent bonds. Besides, cytoplasm of young cells is visualized into more basophilic

color than old cells. Some cellular organelles have basophilic color, for example basophilic granulocytes, basophilic color of agranulocytes cytoplasm (lymphocytes and monocytes). Cellular kern has also basophilic color. Thus the basophilic color coloration indicates the prevalence of anabolic processes into cellular kern, into cytoplasm and especially in cytoplasm of young cells. Some organelles accept more acidic staining agents /for example eosinophilic granulocytes /, i.e. having bonds to positive ionized molecules. These organelles have the other kind of anabolic processes. However the balance of chemical potentials between extracellular medium and intracellular medium should be maintained, depending on the phase of cellular cycle, which is regulated by driving mechanism of cellular transport of substances, according Theorell formula [3, 4, 7]. Unlike the norm there are the unbalanced states between extracellular medium and intracellular medium in cancer tissue because of huge anabolic processes with suppression of catabolic processes and consumption of Acetyl-CoA and energy for anabolic processes that leads to overload of the "nodal point bifurcation of anabolic and catabolic processes" [NPBab] with lack of Acetyl-CoA, causing irrepressible cancer growth and metastasis [7]. Besides the prevalence of anabolic processes promotes as development of cellular cycle or as well as the syntheses of enzymes, antibodies, hormones and the other biologic active substances.

### *2.5. Biophysical Model of the Cellular Mechanisms Promoting Device of Cellular Capacitors*

The cellular wall consists of the external membrane and the internal membrane. All cells have the negative charge on the external membrane on cellular wall [3, 13, 14]. The cause of this charge on a cellular external membrane is the terminal carboxyl group of n-acetyl-neuraminic acid [13, 14]. It is known, according to the Glansdorff and Prigogine theory [15], that the fluctuations of an entropy promote development an open non equilibrium thermodynamic system: The positive fluctuations local production of an entropy ($\Delta_x\beta > 0$) corresponds to cell develops upon the linear Stationary graph, but on the contrary the excessive negative fluctuations of local production an entropy ($\Delta_x\beta < 0$) leads to non-linear Quasi-stationary pathologic development [15]. The moderate negative fluctuations local production of entropy ($\Delta_x\beta < 0$) causes these changes of cell development in norm. Intracellular medium touches on an internal membrane, causing the charge relevant to chemical potential of intracellular medium. Extracellular medium touches on an external membrane, causing the charge relevant to chemical potential of extracellular medium. Thus the difference of charges ($\Delta q$) between an internal membrane and an external membrane of cellular walls is formed.

There are the dielectric layers of proteins between the internal membrane and the external membrane. All of it gives us possibility to make the following conclusion: Functioning membrane is analogous to functioning capacitor. The difference of charges ($\Delta q$) on an internal membrane and an external membrane of a cell promotes an attraction of the ionized molecules to the positive charge of membrane and the ions to the negative charge of membrane.

Thus it is happened the locomotion of ions and ionized molecules to the membranes charges in this phase. The ionized molecules adsorb to positive charges of polarized pole of cellular external membrane. The ions adsorb to negative charges of polarized pole of cellular internal membrane. It takes place processes an attraction of ionized molecules and ions to the cellular membranes according to the charges of polarized poles and to the difference of electrochemical potentials from inside and outside of the cellular wall. Considering that a cellular wall consists as of the polarizable sections acting in processes endocytosis-exocytosis, and as well as of the non electroconductive hydrophobic lipid sections, it is possible to make conclusion, that a cellular wall consists of the many capacitors, separated one from another by the non electroconductive hydrophobic sections of a cellular wall. Cellular capacitors of a cellular wall do not act identically, possessing the different executing dielectric functions of proteins with various properties (dielectric conductivity /dielectric constant/). Dynamism of binding between α-domain and β-domain in the structure of a Receptor gives us possibility to make conclusion that the directivity of dielectric changes in this cellular capacitor is identified with the mechanism of the variable capacitor action. Hence it is possible specific interactions the Receptor with the various Ligands which have the ionic certain chemical potentials. Just the dynamism action of these dielectrics in the Receptors as the analogue of the variable capacitors promotes dynamic rearrangements to various Ligands (hormones, antigens etc.) depending on condition in an organism [3, 16 – 19].

#### *2.5.1. The Cellular Capacitor Mechanism [3]*

Cellular wall consists of an internal membrane and an external membrane which are charged with the different electrical charges ($q_1$ and $q_2$), having difference of potentials ($\Delta\varphi$) or electric-field strength (U). The dielectric substances (proteins) are between an internal membrane and an external membrane, which cause the all conditions for mechanism of cellular capacitor operation, having the dielectric conductivity /dielectric constant/ ($\varepsilon$). The electric capacity (C) of a cellular capacitor is calculated using such equation:

$$C = q / \Delta\varphi = q / U.$$

Besides, there are the some more equations:

$U = Er$; $C = q / Er$; $\sigma = q / S$; $E = \sigma / \varepsilon_o$

(E –strength of field between cellular membranes, r – distance between cellular membranes, $\sigma$ - surface density of charges on cellular membranes, S – the area of the each membrane, $\varepsilon_o$ - electric constant).

Thus the electric capacity of a cellular capacitor is equal:

$C = \sigma S / Er = E\varepsilon_o S / Er = \varepsilon_o S / r$ .

The equation of a capacitor electric capacity turns into such equation in the presence of a dielectric between membranes:

$C = \varepsilon\varepsilon_o S / r$ , i.e. the electric capacity of a capacitor increases in ε times.

(ε - dielectric conductivity /dielectric constant/)

A cellular wall consists of the many capacitors having different dielectric conductivities (ε) and different capacitors' electric capacity (C) due to different proteins between cellular membranes.

Considering that the electric-field strength on a cellular capacitor changes in time via a sinusoidal graph, the equation of the electric capacity of a cellular capacitor is viewed so:

$$C = q /\Delta\varphi \sin \acute{\omega}t = q /U \sin \acute{\omega}t; \acute{\omega} = 2\pi f$$

(C - the electric capacity of a cellular capacitor, q – electrical charge, Δφ - difference of potentials, U - electric-field strength, ώ - angular frequency, f – frequency, t – time).

The wave functions of cellular capacitors are advanced as linear function of decaying wave according to the equation:

$u = U_n /r$ (u - instantaneous voltage, $U_n$ - range of stress, r - distance)

Thus the big distance (r) induces the small instantaneous voltage (u). Besides, the structures of cellular wall's receptors consist of both α-subunit and β-subunit which are the proteins with the properties of dielectric conductivity. The changes of the positions among these subunits between cellular membranes promote the changes of dielectric conductivity of cellular capacitor's Receptor. Thus the structures of cellular Receptors correspond to the different variable capacitors operations, which adjust themselves to wave functions of the various substances.

#### *2.5.2. The Wave Functions of Substances [3, 20]*

Being grounded on the Schreodinger equation of the method of the molecular orbitals – a linear combination of atomic orbitals (MO LCAO), the wave function of any molecule is determined as the total wave functions of the nuclear orbitals, multiplied by the appropriating weight coefficients:

$$\psi = c_1\varphi_1 + c_2\varphi_2 + \ldots c_n\varphi_n \text{ [3, 20].}$$

(ψ - wave function of a molecule, φ - wave functions of the nuclear orbitals, c - the appropriating weight coefficients)

The wave functions of substances are also advanced as decaying wave of linear function according to the equation:

$u = U /r$ (u - instantaneous voltage, U - range of stress, r - distance)

Thus the big distance (r) induces the small instantaneous voltage (u).

#### *2.5.3. Operations of Cellular Capacitors' Resonance Waves on the Wave Functions of strange Substances for Distance Reactions of the Immune Responses and Hormonal Expression (3)*

Having adopted, that the inductive and capacitive components at the moment of a resonance are equal, resonance frequency is defined by the equations:

$\omega_1 L = 1/\omega_1 C$, where is $\omega_1 = 2\pi f$; Hence here is the equation:

$$C\omega_1^2 L = 1$$

($\omega_1$ – a resonance of an angular frequency, f - a resonance frequency, L – an inductance; C – electric capacity of the cellular capacitor).

Thus the resonance of the angular frequency is raised. Therefore the resonance of cellular capacity with the induced substances contributes to increase of resonance waves of a sinusoidal graph as for cellular metabolism as well as for distance reactions on immune responses and the hormonal expression. The resonance of the wave functions of cellular capacity to the wave functions of substances promotes attraction of cells and substances each toward other that reflects the distance reactions on immune responses and the hormonal expression. Just the free cells in blood use the remote reaction across distance due to cellular capacitors operations realizing function immunity of an organism [3].

## 3. Discussion of the Role of cellular Remote Reaction and Contact Reaction in Maintenance Stability Internal Energy and Internal Medium an Organism

Cells-phagocytes rush into the dirty wound with microbes, forming pus [21 – 24]. However the cells-phagocytes do not direct themselves toward the sterile wound, which was treated by antibiotics or disinfectants. Remote reactions of cells to various allergens are available also in the mechanism allergic expressions of an organism. Immunity of an organism depends also on remote reaction of cells of an organism to various pathological agents. Remote cellular reactions precede contact cellular reactions which destroy substances of strange objects in immune responses. Absence of resistibility of an organism is connected also with absence or insufficiency of remote reaction of cells to various pathological agents. Possible mechanisms of hormonal reactions of an organism depend also on remote reaction of cells to various factors. The disturbances of immune and hormonal systems are observed by cancer treatment with great dosage of cytotoxic drugs as a result of these drugs' negative influence on anabolic processes of an organism, destroying as cancer metabolism and as well as also immune and hormonal activity [25].

### *3.1. Discussion of Role Mitochondrial and Nuclear Functions in Maintenance Stability Internal Energy and Internal Medium Both Cells and an Organism in Norm*

An organism's regulative system causes stability Internal Medium and Internal Energy both an organism and cells of an organism [1, 2]. The maintenance stability of cellular Internal Energy and cellular Internal Medium depends on the mutual influences between the mechanism maintenance stability of an organism and the mechanisms maintenance stability of all cells' cytoplasm. Intracellular chemical potential ($\mu_{intcell}$) of each cell interacts with intracellular chemical potentials ($\mu_{intcell}$) of all cells of an organism via resonance waves of remote reaction due to operations of cellular capacitors [3]. Cellular central mechanism of anabolic processes is located in nucleus, and the cellular central mechanism of catabolic processes is located in mitochondrion. Just the interactions between such resisted systems as nucleus and mitochondria can function due to intermediate mechanism which is the mechanism of mitochondrial DNA [mtDNA]. Mitochondrial DNA is subjected to permanent fissions with its lesion due to permanent ruining effect of oxidized free radicals created by permanent arising of ROS, $H_2O_2$ and superoxide [$O_2^{*-}$] which are mediated by GTPase, dynamin-related protein 1 (Drp 1) [26 – 30]. Also mitochondria are subjected to fission due to mitochondrial factor (Mff), reflecting expression of catabolic oxidative processes [26 – 33]. On the other hand, there are the permanent repairing mechanisms via alkylation and mtDNA ligase activity for permanent fusion of destructing mtDNA preventing mtDNA loss via mtDNA repair and maintenance of copy number, reflecting expression of anabolic reductive processes [26]. Thus dynamics of mtDNA fission / fusion is occurred via oscillation balance catabolic / anabolic processes [26, 31 – 33]. Mitochondrial fusion is mediated by OPA1, Mfn1, Mfn2 proteins which are generated by the genes of the same names [26, 34]. Also nuclear dynamic alternations of the destructive function of nuclear DNA (nDNA) via fragmentation, as shift into catabolic processes, in which the caspase-activated DNase (CAD) is an activator, and the function reparations of nuclear DNA (nDNA), as shift into anabolic processes, which is stimulated by mismatch repair proteins (MMR). Thus these connections between oscillations nucleus and mitochondria induce stable moderate oscillations of cellular chemical potential in cytoplasm ($\mu_{cytopl}$) connecting with rhythms of cellular advances via respiratory rhythm, maintaining cellular stability of Internal Energy and Internal Medium, and also via cellular cycle, promoting cellular development. Also chemical potentials of all cells ($\mu_{cell}$) create mutual influences with chemical potential of an organism ($\mu_{org}$) for maintenance common stability Internal Energy and Internal Medium.

# 5. Conclusions

1. Open non equilibrium non linear thermodynamic system a human organism subjected to thermodynamic laws which some parameters were calculated.
2. There were described the regulatory mechanisms of maintenance stability an open non equilibrium non linear thermodynamic system a human organism.
3. There were described mechanisms maintenance stability Stationary State and Quasi-stationary State of both an organism and its cells.
4. There is discussed the role of cellular capacitors creating remote reactions which transit into contact reaction and contribute to hormonal and immune defensive reactions promoting maintenance stability Internal Energy and Internal Medium of Stationary State an organism.
5. There is discussed the role of mitochondrial functions and nuclear function in maintenance stability Internal Energy and Internal Medium both cells of an organism and an organism in norm.

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# References

[1] Ponizovskiy M.R., The Central Regulation of all Biophysical and Biochemical Processes as the Mechanism of Maintenance Stability of Internal Energy and Internal Medium both in a Human Organism and in cells of an Organism, Modern Chemistry & Application, 2013, 1 (1), doi: 10.4172/mca.1000e101.

[2] Ponizovskiy M.R., The mechanisms maintenance stability Internal Energy and Internal Medium an organism in norm and in quasi-stationary pathologic states, Biochemistry & Physiology, 2013, v. 2 (3), doi:10.4172/2168-9652.1000115.

[3] Ponisovskiy M.R., Driving mechanisms of passive and active transport across cellular membranes as the mechanisms of cell metabolism and development as well as the mechanisms of cellular distance reaction on hormonal expression and the immune response, Critical Reviews in Eukaryotic Gene Expression, 2011, 21 (3), 267 – 290.

[4] Ponizovskiy M.R., Biophysical and biochemical models of cellular development mechanisms via cellular cycle as in normal tissue and as well as in cancer tissue and in inflammatory processes, Critical Reviews in Eukaryotic Gene Expression, 2013, v. 23 (2), 171 – 193.

[5] Ponizovskiy M.R., Biophysical and biochemical transmutation of mitochondrial function in cancer genesis, Biochemistry & Analytical Biochemistry, 2013, v. 2 (3), doi:10.4172/2161-1009.1000137.

[6] Sato Norio, Chemical Energy and Energy: An introduction to chemical thermodynamics for Engineers, 2004, 160.

[7] M. Ponisovskiy, Cancer metabolism and the Warburg effect as anabolic process outcomes of oncogene operation, Critical Reviews in Eukaryotic Gene Expression, v.20 (4), (2010), 325-339.

[8] R. N. Glebov, Endocytosis and exocytosis, 1987, vol. 2, 7-93, in book: A.A.Boldirev (Ed), Biochemistry of membranes, High school, Moscow. [in Russian] [Р.Н.Глебов, Эндоцитоз и Экзоцитоз, 1987, книга 2, 7-93, в: А.А.Болдырев (Ред), Биохимия мембран, Высшая школа, Москва]

[9] A. J. Kulberg, Receptors of cellular membranes, 1987, vol. 4, 5-100, in book: A.A.Boldirev (Ed), Biochemistry of membranes, The high school, Moscow. [in Russian] [А.Я.Кульберг, Рецепторы клеточных мембран, 1987, книга 4, 5-100, в: А.А.Болдырев (Ред), Биохимия мембран, Высшая школа, Москва]

[10] E. A. Ferenczi, J. A. Fraser, S. Chawla, J. N. Skepper, C. J. Schwiening, and C. L. Huang, Membrane potential stabilization in amphibian skeletal muscle fibres in hypertonic solutions, The Journ. of Physiol.", 2004, 555 (2), 423-438.

[11] M. R. Ponizovsky, V. A Kalibabchuk., V. A. Samarsky, A. V. Tofan, A. A. Orlovsky, Ponizovska T. M., "The thermodynamic conception of system of metabolic processes and its possible application to pathology", "Actual problems of medicine and biology", Kiev, 2000, №1, p.232 – 245. [in Russian] [М.Р.Понизовский, В.А.Калибабчук, В.А.Самарский, А.В.Тофан, А.А.Орловский, Понизовская Т.М., «Термодинамическая концепция системы метаболических процессов и её возможные приложения к патологии», «Актуальные проблемы медицины и биологии», Киев, 2000, №1, с.232 – 245]

[12] T. Wileman, C. Harding, Ph. Ftahl, Receptor-mediated endocytosis, Biochem. J., 1985, vol.232, 1-14.

[13] G. Ruhenstroth-Bauer, Experiments with Electrophoresis of cells, Library hematology, 1960, v.12, 5-19. [in German] [G.Ruhenstroth-Bauer, Erfahrungen bei der Elektrophorese von Blutzellen, Bibl. haemat., 1960, v.12, 5-19]

[14] A. Zerial and D. J. Wilkins, Electrophoretic Behaviour of some Human Blood Cells, Cellular and Molecular Life Sciences (CMLS), 1972, v.28, 1435-1436.

[15] P. Glansdorff and I. Prigogine, Thermodynamic Theory of Structure, Stability, and Fluctuations, Wiley, London, 1971, p.306.

[16] R. L. P. Adams, Cell Culture for biochemists, Elsevier / North–Holland Biomedical Press Amsterdam New York Oxford, 1980, 7-256.

[17] R.V. Petrov, R. I. Ataullahanov, Cellular membranes and immunity, in: A. A. Boldirev (Ed), Biochemistry of membranes, The high school, Moscow, 1991, vol. 9, 1-139. [in Russian] [Р.В.Петров, Р.И.Атауллаханов, Клеточные мембраны и иммунитет, в: А.А.Болдырев (Ред), Биохимия мембран, Высшая школа, М., 1991, книга 9, 1-139]

[18] A.J.Kulberg, Molecular immunology, The science "Medicine", Moscow, 1985, 1-287.[in Russian] [А.Я.Кульберг, Молекулярная иммунология, Наука «Медицина», Москва, 1985, 1-287]

[19] A.J.Kulberg, Regulation of immune response, Medicine, Moscow, 1986, 1-223. [in Russian] [А.Я.Кульберг, Регуляция иммунного ответа, Медицина, Москва, 1986, 1-223]

[20] Francis A.Carley and Richard J.Sundberg, Advanced organic chemistry, Part A: Structure and Mechanisms, 1983, 671p.

[21] G.P.Lewis, Mediators of Inflammation, Inflammation Research, Birkhäuser Basel, 1986, vol. 19, №1-2, 55-56.

[22] B.D.Brondz, T-lymphocytes and their receptors in immunologic recognition, Science, Moscow, 1987, 13-17. [in Russian] [Б.Д.Брондз, Т-лимфоциты и их рецепторы в иммунологическом распознавании, Наука, Москва, 1987, 13-17]

[23] M.K.L.Collins, M.J.Owen, The T-cell antigen receptor, Biochem. J., 1985, v.230, 281-291.

[24] K.E.Mostov, M.Friedlander, G.Blobel, The receptor for transepithelial transport of IgA and IgM contains multiple immunoglobulin-like domains, Nature, 1984, v.308, 37-43.

[25] M.Ponisovskiy, Warburg effect mechanism as the target for theoretical substantiation of a new potential cancer treatment, Critical Reviews in Eukaryotic Gene Expression, 2011, vol.21 (1), 13-28.

[26] Furda Amy Marie, The role of mtDNA damage in mitochondrial dysfunction, University of Pittsburg (defended dissertation 2011), 2011, 145p.

[27] Tedesco A.C., Martínez L. and González S., Photochemistry and photobiology of actinic erythema: defensive and reparative cutaneous mechanisms, Braz. J. Med. Biol. Res., 1997, v. 30(5) 561 – 575.

[28] Frohe L., Free radicals in biology, in Pryor W.A. (ed), Academic Press, New York, 1982, 223 – 275.

[29] Rhee S.G., Cell signaling. $H_2O_2$, a necessary evil for cell signaling, Science, 2006, 312, 1882 – 1883.

[30] Lambert A.J. and Brand M.D., Reactive oxygen species production by mitochondria, Methods Mol. Biol., 2009, 554, 165 – 181.

[31] Westermann B., Mitochondrial fusion and fission in cell life and death, Nat. Rev. Mol. Cell Biol., 2010, 11, 872 – 884.

[32] Hales K.G. and Fuller M.T., Developmentally regulated mitochondrial fusion mediated by a conserved, novel, predicted GTPase, Cell, 1997, 90, 121 – 129.

[33] Meeusen S., McCaffery J.M. and Nunnary J., Mitochondrial fusion intermediates revealed in vitro, Science, 2004, 305, 1745 – 1752.

[34] Olichon A., Emorine L.J., Decoins E. et al., The human dynamin-related protein OPA 1 is anchored to the mitochondrial inner membrane facing the inter-membrane space, FEBS Lett., 2002, 523, 171 – 176.